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Original Research Paper

Neurology

A CASE OF IDIOPATHIC INTRACRANIAL HYPOTENSION PRESENTED AS DIPLOPIA

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ABSTRACT

Introductions Dysfunction of sixth cranial nerve can give symptom of horizontal diplopia and can result from lesion occuring anywhere along its course from pons to orbit. Bilateral involvement suggest a diffuse process such as changes in intracranial tension and sometimes make it difficult to localize the lesion **Purpose** To report bilateral sixth nerve palsy as initial presentation of spontaneous intracranial hypotension in a young patient **Case Report** A 16 year old female presented with binocular horizontal diplopia that persist all day since 1 week associated with significant headache and 1 episode of vomiting. She was denying any history of trauma, loss of consciousness, nor hematological coagulopathy. The examination found her eyes position was esotropia 7° bilateral, with limited eye movements in both eyes to the lateral sides. Fundoscopic examination shows bilateral normal optic disc. Mri brain with angiography shows type 1 vascular loop of anterior inferior cerebral artery in internal auditory meatus with pachymeningeal enhancement. On csf opening pressure measurement it was 50mm of water. Patient was treated with hydration and bed rest and fully recovered after 3 weeks. **Conclusion** The sixth cranial nerve palsy with intracranial hypotension without any trauma or coagulopathy suggest spontaneous intracranial hypotension for which pt should be treated with hydration and bed rest with evaluation of brain by mri for any visible leak. If no any visible defect seen and patient is not improving from conservative management she should be undergo myelography or radionuclide ciesternography.

KEYWORDS : Bilateral Sixth Nerve Palsy, Intracranial Hypotension, Postural Headache

1. INTRODUCTION

The sixth cranial nerve (CN VI) innervates the ipsilateral lateral rectus, which function to abduct the ipsilateral eye. Patients with CN VI palsy of any causes mostly come to the ophthalmologist first because of binocular horizontal diplopia that worse in the field of action of the paretic lateral rectus muscle. The causes of acquired CN VI palsy are different depending on the age of the patient. Common causes in young adult are central nervous system mass, demyelinating disease or idiopathic causes. The sixth cranial nerve palsy may occur unilaterally and bilaterally. Bilateral Involvement suggest a diffuse process such as midline mass lesion that extends bilaterally, a meningeal-based process or changes in intracranial pressure.

Dysfunction of CN VI can result from lesion occuring anywhere along its course between the CN VI nucleus in the dorsal pons and the lateral rectus muscle within the orbit. Prompt and correct diagnosis by ophthalmologist is critical in determining the causes and, therefore, proper evaluation and treatment can be done. In this case we will discuss the work up of bilateral sixth nerve palsy in a young patient with Spontaneous intracranial hypotension.

2. Case Report

A 16 year old female came to G.G.Hospital with a chief complaint of double vision that persist all day since 1 week. The patient reported the double vision had begun gradually. The diplopia was mostly at distance but was occasionally noted at near. Separation of object was horizontal. The patient had to closed one eye in order to relieve diplopia. The patient had history of severe headache 1 week ago associated with nausea and vomiting of 1 episode where headache is mostly in upright position and decrease intensity with lying down. She was denying any history of traumatic brain injury or did any high intensity sports. There was no history of loss of consciousness, seizure, slurred speech, extremities weakness, fever, malaise, or prolonged cough. She did not smoke nor consume alcohol. She had no any previous history of anticoagulant therapy or hematological coagulopathy, medication of tuberculosis and was in good health before this episode. The patient was alert and fully oriented. On physical examination reveled the vital signs were normal tone, power, reflexes, sensation of the four limbs. Opthalmology examination reveled her visual acuity of the right eye was 1,0 and the left eye was 0,4 ph 0,63. The eye position was esotropia 7°, with limited eye movements in both eyes to the lateral sides. There wasn't any nystagmus.

Anterior segment examination was within normal limits in both eyes. Fundoscopic examination is normal in both eyes. Color perception test, amsler, and contrast reading test were within normal limit in both eyes. Patient has no facial paralysis and corneal sensitivity was normal.

In Initial mri brain with orbit shows no any abnormalities. After 4 days again mri brain with angiography done which shows pachymeningeal enhancement with grade 1 vascular loop of anterior inferior cerebral artery in internal auditory meatus.

On csf examination a non traumatic lumbar puncture was made and with help of manometer the opening pressure recorded was 50mm of water compatible with low csf pressure. Csf examination shows cells 3-4 leucocytes/dl, csf protein were 45mg/dl, csf glucose was 44 mg/dl.

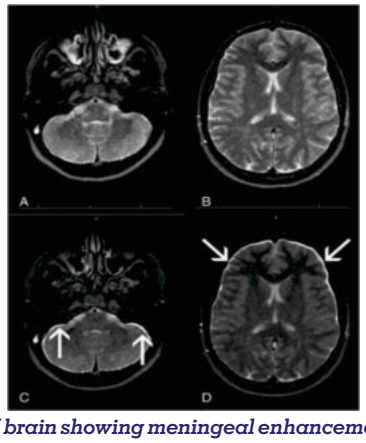

Fig.1 – MRI brain showing meningeal enhancement

The patient was diagnosed with bilateral sixth nerve palsy due to spontaneous intracranial hypotension. She was given eye patch with intravenous fluid support of normal saline and

dextrose normal saline for 5 days. After that she had improvement in diplopia and after 1 month she was completely asymptomatic.

3. DISCUSSION

It is important to perform a careful evaluation of the occular motility when a patient present with a complaint of new onset binocular diplopia, including a cover test in all positions of gaze. A complaint of horizontal diplopia, worse at distance and in one direction of lateral gaze, is consistent with an abduction deficit and presents as an esodeviation, with the greatest esodeviation in the direction of the abduction deficit.

Once an abduction deficit is noted, the etiology must then be determined. Possible etiologies for an abduction deficit include problems with the extraocular muscles, neuromuscular junction, CN VI, and pons. In terms of the extraocular muscles, this could be either damage/trauma to the lateral rectus muscle or enlargement/ infiltration of the medial rectus muscle, such as in Graves's disease. This could also be restriction from an orbital mass. A restrictive process would exhibit a positive forced duction test. In terms of the neuromuscular junction, this would be associated with myasthenia gravis and could present with any motility abnormality, as well as with ptosis. A pontine lesion could also present with contralateral weakness caused by crossing of the corticospinal tract. Because of the proximity of cranial nerve VI and cranial nerve VII in the pons, the function of cranial nerve VII should be assessed to try to localize the lesion.

If a CN VI palsy is suspected, it should be determined if there are any localizing features to indicate where along the route of the CN VI the injury has occurred. Once it exits the pons, CN VI enters the subarachnoid space, where it runs along the bony Clivus, until it enters Dorello's canal, where it is firmly anchored. From Dorello's Canal, CN VI enters the cavernous sinus, where it travels more medially to enter the orbit through the superior orbital fissure and then travels laterally in the orbit to innervate the lateral rectus muscle. A good knowledge of the pathway of CN VI can

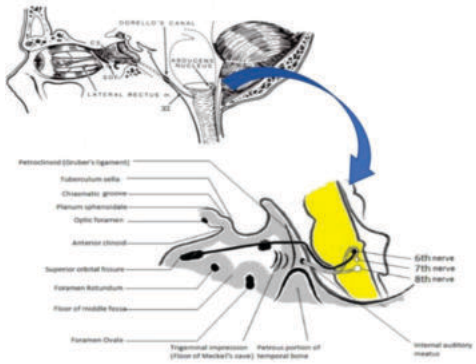

Fig.2 – Lateral view of the cranial nerve VI.

assist in identifying the location of the lesion and in the differential diagnosis of CN VI palsy. Spontaneous intracranian hypotension (SIH) is a rare but well known entity, described for the first time by Schaltenbrand in 1938 and actually classified in the International Classification of Headache Disorders 2nd Edition (ICHD-II) in 2004.

The clinical picture is very heterogeneous, including a not always orthostatic headache that may be associated with one or more several other Symptoms, including pain or stiffness of the neck, nausea,Horizontal diplopia, dizziness, visual blurring and photophobia. The diagnosis of SIH is generally one of exclusion, so that precipitating events such as recent operative procedures, lumbar puncture or traumatic dural tear and other medical conditions (severe systemic infection, diabetic coma, administration of hypertonic solution) must be ruled out.

The most frequent symptom is orthostatic headache that worsening within 15 minutes after adopts an upright position and improves after lying down. Nonetheless, it is not unusual to achieve the diagnosis in patients with atypical symptomatology as no headache or other types of headache, cranial nerve palsies, coma, parkinsonism, ataxia, chorea, unilateral hearing loss or cerebellar hemorrhage. Image findings are relevant to suspect this entity or to confirm the diagnosis. In cranial MRI we can observe pachymeningeal enhancement, sagging of the brain, engorgement of venous structures and subdural fluid collections. MRI mielography is the gold standard imaging test for the diagnosis of spontaneous intracranial hypotension due to ability to localize CSF leak with sensitivity of approximately 89%.

In our case, the woman debuted with right VI cranial nerve palsy that produced horizontal diplopia and orthostatic headache. Cranial nerve palsies can be explained by the displacement of the brain after CSF lost and subsequently the stretching of the nerves.

Conservative treatment consisting of bed rest, adequate fluid intake and oral caffeine, is the first choice of treatment. If this approach fails, epidural blood patch application is an alternative. Surgical repair of CSF leak is the last alternative of treatment only reserved for those patients in whom non-surgical treatments have failed.

4. CONCLUSION

In conclusion, SIH is a syndrome that we have to consider in patients with atypical headache or with typical image findings. Other symptoms including drowsiness and altered mental status cannot be overlooked.

5. Conflict Of Intrest

None

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